

Training Since 1992...2001

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ATA INTERNATIONAL LOGOFF AT 161141Z ON 19 AUG 1991

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FILE 'MULLINE' ENTERED AT 161042Z ON 10 AUG 81

Table 1. *Salmonella* serotypes and phage types isolated from the 1997-1998 salmonellosis outbreak in the Netherlands

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Table 1. *Salmonella* serotypes and their associated diseases. The table lists the serotypes and the diseases they are associated with, categorized by the type of disease (Enteric fever, Enteric fever-like illness, and Enteric fever-like illness with systemic manifestations).

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Figure 1. The effect of the concentration of the *Ag* on the *Ag* adsorption capacity of the *Ag*-*Ag*2O-*Ag*2S-*Ag*2Se-*Ag*2Te-*Ag*2I-*Ag*2Br-*Ag*2Cl-*Ag*2S2O3-*Ag*2S2O4-*Ag*2S2O5-*Ag*2S2O6-*Ag*2S2O7-*Ag*2S2O8-*Ag*2S2O9-*Ag*2S2O10-*Ag*2S2O11-*Ag*2S2O12-*Ag*2S2O13-*Ag*2S2O14-*Ag*2S2O15-*Ag*2S2O16-*Ag*2S2O17-*Ag*2S2O18-*Ag*2S2O19-*Ag*2S2O20-*Ag*2S2O21-*Ag*2S2O22-*Ag*2S2O23-*Ag*2S2O24-*Ag*2S2O25-*Ag*2S2O26-*Ag*2S2O27-*Ag*2S2O28-*Ag*2S2O29-*Ag*2S2O30-*Ag*2S2O31-*Ag*2S2O32-*Ag*2S2O33-*Ag*2S2O34-*Ag*2S2O35-*Ag*2S2O36-*Ag*2S2O37-*Ag*2S2O38-*Ag*2S2O39-*Ag*2S2O40-*Ag*2S2O41-*Ag*2S2O42-*Ag*2S2O43-*Ag*2S2O44-*Ag*2S2O45-*Ag*2S2O46-*Ag*2S2O47-*Ag*2S2O48-*Ag*2S2O49-*Ag*2S2O50-*Ag*2S2O51-*Ag*2S2O52-*Ag*2S2O53-*Ag*2S2O54-*Ag*2S2O55-*Ag*2S2O56-*Ag*2S2O57-*Ag*2S2O58-*Ag*2S2O59-*Ag*2S2O60-*Ag*2S2O61-*Ag*2S2O62-*Ag*2S2O63-*Ag*2S2O64-*Ag*2S2O65-*Ag*2S2O66-*Ag*2S2O67-*Ag*2S2O68-*Ag*2S2O69-*Ag*2S2O70-*Ag*2S2O71-*Ag*2S2O72-*Ag*2S2O73-*Ag*2S2O74-*Ag*2S2O75-*Ag*2S2O76-*Ag*2S2O77-*Ag*2S2O78-*Ag*2S2O79-*Ag*2S2O80-*Ag*2S2O81-*Ag*2S2O82-*Ag*2S2O83-*Ag*2S2O84-*Ag*2S2O85-*Ag*2S2O86-*Ag*2S2O87-*Ag*2S2O88-*Ag*2S2O89-*Ag*2S2O90-*Ag*2S2O91-*Ag*2S2O92-*Ag*2S2O93-*Ag*2S2O94-*Ag*2S2O95-*Ag*2S2O96-*Ag*2S2O97-*Ag*2S2O98-*Ag*2S2O99-*Ag*2S2O100-*Ag*2S2O101-*Ag*2S2O102-*Ag*2S2O103-*Ag*2S2O104-*Ag*2S2O105-*Ag*2S2O106-*Ag*2S2O107-*Ag*2S2O108-*Ag*2S2O109-*Ag*2S2O110-*Ag*2S2O111-*Ag*2S2O112-*Ag*2S2O113-*Ag*2S2O114-*Ag*2S2O115-*Ag*2S2O116-*Ag*2S2O117-*Ag*2S2O118-*Ag*2S2O119-*Ag*2S2O120-*Ag*2S2O121-*Ag*2S2O122-*Ag*2S2O123-*Ag*2S2O124-*Ag*2S2O125-*Ag*2S2O126-*Ag*2S2O127-*Ag*2S2O128-*Ag*2S2O129-*Ag*2S2O130-*Ag*2S2O131-*Ag*2S2O132-*Ag*2S2O133-*Ag*2S2O134-*Ag*2S2O135-*Ag*2S2O136-*Ag*2S2O137-*Ag*2S2O138-*Ag*2S2O139-*Ag*2S2O140-*Ag*2S2O141-*Ag*2S2O142-*Ag*2S2O143-*Ag*2S2O144-*Ag*2S2O145-*Ag*2S2O146-*Ag*2S2O147-*Ag*2S2O148-*Ag*2S2O149-*Ag*2S2O150-*Ag*2S2O151-*Ag*2S2O152-*Ag*2S2O153-*Ag*2S2O154-*Ag*2S2O155-*Ag*2S2O156-*Ag*2S2O157-*Ag*2S2O158-*Ag*2S2O159-*Ag*2S2O160-*Ag*2S2O161-*Ag*2S2O162-*Ag*2S2O163-*Ag*2S2O164-*Ag*2S2O165-*Ag*2S2O166-*Ag*2S2O167-*Ag*2S2O168-*Ag*2S2O169-*Ag*2S2O170-*Ag*2S2O171-*Ag*2S2O172-*Ag*2S2O173-*Ag*2S2O174-*Ag*2S2O175-*Ag*2S2O176-*Ag*2S2O177-*Ag*2S2O178-*Ag*2S2O179-*Ag*2S2O180-*Ag*2S2O181-*Ag*2S2O182-*Ag*2S2O183-*Ag*2S2O184-*Ag*2S2O185-*Ag*2S2O186-*Ag*2S2O187-*Ag*2S2O188-*Ag*2S2O189-*Ag*2S2O190-*Ag*2S2O191-*Ag*2S2O192-*Ag*2S2O193-*Ag*2S2O194-*Ag*2S2O195-*Ag*2S2O196-*Ag*2S2O197-*Ag*2S2O198-*Ag*2S2O199-*Ag*2S2O200-*Ag*2S2O201-*Ag*2S2O202-*Ag*2S2O203-*Ag*2S2O204-*Ag*2S2O205-*Ag*2S2O206-*Ag*2S2O207-*Ag*2S2O208-*Ag*2S2O209-*Ag*2S2O210-*Ag*2S2O211-*Ag*2S2O212-*Ag*2S2O213-*Ag*2S2O214-*Ag*2S2O215-*Ag*2S2O216-*Ag*2S2O217-*Ag*2S2O218-*Ag*2S2O219-*Ag*2S2O220-*Ag*2S2O221-*Ag*2S2O222-*Ag*2S2O223-*Ag*2S2O224-*Ag*2S2O225-*Ag*2S2O226-*Ag*2S2O227-*Ag*2S2O228-*Ag*2S2O229-*Ag*2S2O230-*Ag*2S2O231-*Ag*2S2O232-*Ag*2S2O233-*Ag*2S2O234-*Ag*2S2O235-*Ag*2S2O236-*Ag*2S2O237-*Ag*2S2O238-*Ag*2S2O239-*Ag*2S2O240-*Ag*2S2O241-*Ag*2S2O242-*Ag*2S2O243-*Ag*2S2O244-*Ag*2S2O245-*Ag*2S2O246-*Ag*2S2O247-*Ag*2S2O248-*Ag*2S2O249-*Ag*2S2O250-*Ag*2S2O251-*Ag*2S2O252-*Ag*2S2O253-*Ag*2S2O254-*Ag*2S2O255-*Ag*2S2O256-*Ag*2S2O257-*Ag*2S2O258-*Ag*2S2O259-*Ag*2S2O260-*Ag*2S2O261-*Ag*2S2O262-*Ag*2S2O263-*Ag*2S2O264-*Ag*2S2O265-*Ag*2S2O266-*Ag*2S2O267-*Ag*2S2O268-*Ag*2S2O269-*Ag*2S2O270-*Ag*2S2O271-*Ag*2S2O272-*Ag*2S2O273-*Ag*2S2O274-*Ag*2S

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2  1.4e9 ANTIBODY 1.0N INTERFERON GAMMA IF IFN GAMMA IF GAMMA
3  INTERFERON OR GAMMA IFN

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# 2. antibodies: TNF or TNF alpha or tumor necrosis factor alpha or TNF beta or tumor necrosis factor beta
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FEDERAL BUREAU OF INVESTIGATION

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Figure 1. Schematic representation of the experimental design. The subjects were divided into two groups: the control group (C) and the experimental group (E). The control group (C) was divided into two subgroups: the control group (C) and the control group (C). The experimental group (E) was divided into two subgroups: the experimental group (E) and the experimental group (E). The control group (C) was divided into two subgroups: the control group (C) and the control group (C). The experimental group (E) was divided into two subgroups: the experimental group (E) and the experimental group (E).

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AB Organisms belonging to the Mycobacterium avium complex (MAC) are the most common bacterial pathogens in patients with AIDS but factors associated with the activation of cellular defense mechanisms against this atypical mycobacterium have not been defined. Peritoneal macrophages harvested from a chronic MAC infection in C57 black mice are able to kill approximately 40% of intracellular MAC in contrast to 10-20% killing by stimulated human and mouse macrophages in vitro. The availability of human IFN-gamma, rIFN-gamma, and rIL-2 permitted evaluation of the role of each of these lymphokines in activating macrophages in vitro. In combination, rIFN-gamma and rIL-2 stimulated human macrophages to kill MAC. Human macrophage-derived macrophages were cultured in vitro, stimulated with rIL-2, rIFN-gamma, or rTNF, and then infected with MAC serovars 1 and 4. Mouse peritoneal macrophages were harvested, cultured in vitro, and stimulated with rIFN-gamma. rIFN-gamma was associated with a modest increase in intracellular killing of MAC (56% vs 50% even when utilized 24 or 48 h after macrophage infection or when administered for 5 consecutive days after infection). Both human and murine IFN-gamma were associated with increased intracellular growth of MAC (56% vs 40% for murine and 56% vs 40% for human macrophages). However, intracellular killing of MAC was improved with rIL-2 compared with control. This latter effect was fully blocked by anti-TNF antibody, whereas rIL-2 alone did not augment the intracellular killing of MAC by human macrophages. rTNF plus either rIFN-gamma or rIL-2 increased intracellular killing of MAC, but subsequent MAC killing was no greater than with rTNF alone. Treatment of macrophages with 10 U/ml of rIFN followed by rIL-2 (10 U/ml) was associated with 60% intracellular killing. TNF seems to be an important monokine, promoting activation of mycobactericidal mechanisms in human macrophages.

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and alleviated splenomegaly and hypernatraemic polyuria, precursors of signs of development of acquired immune deficiency syndrome (AIDS). These findings could provide insight into the roles of immunomodulator in AIDS treatment as well as the mechanisms by which retrovirus infection induces cytokine dysregulation, facilitating immunodeficiencies in AIDS.

11 Anti-IL-4 monoclonal antibody and IFN-gamma administration retard development of immune dysfunction and cytokine dysregulation during murine AIDS

AB This study was designed to evaluate administration of anti-interleukin-4 (anti-IL-4) monoclonal antibody (mAb), interferon gamma (IFN-gamma), and their combination after LP-PM retrovirus infection of female C57BL/6 mice would prevent retrovirus-induced immunosuppression and cytokine dysregulation. Splenic natural killer (NK) cell activity, T- and B-cell proliferation, and T-helper type 1 (Th1) and Th2 cytokines (IL-2, IFN-gamma, IL-6 and IL-10, and monokine (IL-6 and tumor necrosis factor-alpha (TNF-alpha)) secretions were monitored, as they are usually altered dramatically after murine retrovirus infection. Administration of IFN-gamma and anti-IL-4 significantly prevented retrovirus-induced suppression of splenic NK cell activity, and splenic T- and B-cell proliferation. They also significantly slowed retrovirus-induced elevation of Th2 cytokine (IL-6 and IL-10) release and monokine (IL-6 and TNF-alpha) secretion by splenocytes. They prevented the loss of Th1 cytokine (IL-2 and IFN-gamma) release by splenocytes, and alleviated splenomegaly and hypernatraemic polyuria, precursors of development of acquired immune deficiency syndrome (AIDS). These findings could provide insight into the roles of immunomodulator in AIDS treatment as well as the mechanisms by which retrovirus infection induces cytokine dysregulation, facilitating immunodeficiencies in AIDS.

87 AIDS monoclonal antibody interferon gamma interferon

17 Antineoplasms

PL: BAC (B) Biological activity of effector, except adverse ; B1 L

Biological study

anti-interleukin 4 monoclonal antibody and IFN-gamma retardation of immune dysfunction and cytokine dysregulation during murine AIDS

17 Lymphokines and Cytokines

PL: ALV (Adverse effect, including toxicity) ; MFM (Metabolic formation) ;

B10L (Biological study) ; FORM (Formation, nonpreparative)

cytokine (interleukin 4) and anti-interleukin 4 monoclonal antibody and IFN-gamma administration in murine AIDS effect on formation of

17 Spleen, disease

hyperplasia, anti-interleukin 4 monoclonal antibody and IFN-gamma administration in murine AIDS effect on

17 Lymphokines and Cytokines

PL: BAC (B) Biological activity of effector, except adverse ; B10L

Biological study

(interleukin 4, anti-interleukin 4 monoclonal antibody and IFN-gamma) retardation of immune dysfunction and cytokine dysregulation during murine AIDS

17 Lymphokines and Cytokines

PL: ALV (Adverse effect, including toxicity) ; MFM (Metabolic formation) ;

B10L (Biological study) ; FORM (Formation, nonpreparative)

interleukin 4, anti-interleukin 4 monoclonal antibody and IFN-gamma administration in murine AIDS effect on formation of

17 Lymphokines and Cytokines

PL: ALV (Adverse effect, including toxicity) ; MFM (Metabolic formation) ;

B10L (Biological study) ; FORM (Formation, nonpreparative)

interleukin 4, anti-interleukin 4 monoclonal antibody and IFN-gamma administration in murine AIDS effect on formation of

17 Lymphokines and Cytokines

PL: ALV (Adverse effect, including toxicity) ; MFM (Metabolic formation) ;

B10L (Biological study) ; FORM (Formation, nonpreparative)

tumor necrosis factor-alpha, anti-interleukin 4 monoclonal antibody and IFN-gamma administration in murine AIDS

effect on formation of

17 Interferons

PL: BAC (B) Biological activity of effector, except adverse ; B10L

Biological study

gamma, anti-interleukin 4 monoclonal antibody and IFN-gamma, retardation of immune dysfunction and cytokine dysregulation during murine AIDS

17 Antineoplasms

PL: ALV (Adverse effect, including toxicity) ; MFM (Metabolic formation) ;

B10L (Biological study) ; FORM (Formation, nonpreparative)

gamma, metabolic disorders, hypernatraemic polyuria, anti-interleukin 4 monoclonal antibody and IFN-gamma administration in murine AIDS effect on

20 115 115

FILE 'HOME' ENTERED AT 16:02:46 ON 19 AUG 2001

FILE 'MELINE, CAPLOW, EMBASE, RIOSIS' ENTERED AT 16:23:06 ON 19 AUG 2001

11 1449 S ANTIBODY (IL-4) INTERFERON GAMMA OR IFN GAMMA OR GAMMA
12 1437 S ANTIBODY (IL-4) (TNF) OR (TNF ALPHA) OR TUMOR NECROSIS
13 937 S ANTIBODY (IL-4) (TNF ALPHA) OR TUMOR NECROSIS FACTOR ALPHA
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FILE '115 115' ENTERED AT 16:02:46 ON 19 AUG 2001

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13 937 S ANTIBODY (IL-4) (TNF ALPHA) OR TUMOR NECROSIS FACTOR ALPHA
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ALL INFORMATION CONTAINED
HEREIN IS UNCLASSIFIED
DATE 11-17-2014 BY 60324
AUTHORITY: 105-101-10000
105-101-10000
105-101-10000

T1: Anti-IL-4 monoclonal antibody and IFN- γ administration retards development of immune dysfunction and cytokine dysregulation during murine AIDS

AIDS T cell loss, HIV-1, interleukin 4 interferon
 II Antiretroviral
 BA = "Bacterial activity", as affected, except adverse / BIL
 Biological study
 antihistaminic & monoclonal antibody and IFN-gamma, retardation of
 immune dysfunction and cytokine dysregulation during murine
AIDS

hyperplasia, anti-interleukin 4 monoclonal antibody and IFN- γ administration in murine AIDS effect on

[illegible]

10 tumor necrosis factor α ,
11 interferon- γ antibody and IFN-
12 γ administration in murine AIDS
13 effects on lymphoid

7. Disinfectants, Environmental stresses
 FULMICV Adverse effect, including toxicity; BIL BIL oral study
 gamma, retarding bacteria, hypergamma bacteremia,
 anti-infective gamma oral study by anti-IPN-gamma, administered in
 in Human AIDS effect on

[illegible]

1. *Journal of the American Medical Association*, 1997; 277: 1033-1036.

1. ANSWER 1: P
 APPLICATION NUMBER: 1999-0111
 DOCUMENT NUMBER: 19 011111
 TITLE: Treatment of autoimmune diseases, including AIDS
 INVENTOR S: Skurkovich, Boris; Skurkovich, Simon V.
 PATENT ASSIGNEE S: Advanced Biotherapy Concepts, Inc., USA
 ADDRESS: 10111, 10111, Subt.-in part of U.S. 5,777,543.
 PAREN: USANAM
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY APP. NO. COUNT:
 PATENT INFORMATION:

[illegible]

A5 The present disclosure concerns the treatment of a patient with autoimmune disease, including AIDS, by neutralizing, removing or inhibiting different types of interferons, tumor necrosis factor, HLA class II antigens, IgE, and other pathol. factors and/or their receptors, as well as neutralizing, removing or inhibiting autoantibodies, including antibodies to target cells, CD4 cells and DNA. Treatment comprises administration of an autoimmune inhibitor, or extracorporeal exposure of the patient's fluid to an immunosorbent comprising an autoimmune inhibitor, followed by return of the treated fluid to the patient, or it comprises a combined therapy involving extracorporeal immunosorption in combination with the administration of an autoimmune inhibitor. Combination of a plurality of two or more components selected from anti-interferon, interferon receptor, anti-tumor necrosis factor or receptor, anti-HLA class II receptor, anti-tumor necrosis factor or receptor, anti-IgE, interferon γ receptor, anti-tumor necrosis factor or receptor, and anti-interleukin antibodies are disclosed.

REFERENCE COUNT: 21
 REFERENCE S.S.: 1 Alergia Arthritis Rheum 1991, Vol 4, 1111
 MEDLINE
 2 Alergia W 1991;10:1-11
 3 Alergia W 1991;10:1-11
 4 Alergia W 1991;10:1-11
 5 Alergia W 1991;10:1-11
 6 Alergia W 1991;10:1-11
 7 Alergia W 1991;10:1-11
 8 Alergia W 1991;10:1-11
 9 Alergia W 1991;10:1-11
 10 Alergia W 1991;10:1-11
 11 Alergia W 1991;10:1-11
 ALL INFORMATION AVAILABLE IN THE BB FORMAT

Department of Immunology, University of Medicine and Pharmacy, Bucharest, Romania
Skurkovich, Boris; Skurkovich, Simon V.
 The present invention describes the treatment of a patient with an immunodeficiency disease, including **AIDS**, by neutralizing, removing or inhibiting different types of interfering, tumor necrosis factor, HLA class II antigens, IgE, and other pathologic factors and/or their receptors, as well as neutralizing, removing or inhibiting autoantibodies, including antibodies to target cells, T4 cells and RNA. Treatment comprises administration of an autoimmune inhibitor, or exoantiserum. Exposure of the patient's fluid to an immunosorbent comprising an autoimmune inhibitor, followed by return of the treated fluid to the patient, or it comprises a combined therapy involving exoantiserum immunosorption in conjunction with the administration of an autoimmune inhibitor. Combination of a plurality of two or more components selected from anti-alpha interferon or receptor, anti-beta interferon or receptor, anti-gamma interferon or receptor, anti-tumor necrosis factor or receptor, and anti-interleukin-2 antibodies are disclosed.

- AIDS disease
- Ankylosing spondylitis
- Autoimmune disease:
- Celiac's syndrome
- Crohn's disease
- Graft rejection systems
- Extraintestinal inflammation
- Insulin dependent diabetes mellitus
- Leprosy
- Multiple sclerosis
- Pathogen
- Psoriasis
- Rheumatoid arthritis
- Sarcoidosis
- Systemic lupus erythematosus
- Testis
- Transplant rejection
- Tuberculosis
- Tuberculosis, leprosy, and other diseases: significant effects on normal intestinal flora and on the immune system, and on the development of the immune system and on the immune system.

[illegible]

PROPERTY APPL. INFO.:

| | | |
|-----------------|----|----------|
| US 1996-771431 | A | 19961223 |
| US 1996-25456 | A2 | 19930226 |
| WO 1997-US24266 | W | 19971222 |

AR The present disclosure concerns the treatment of a patient with autoimmune disease, including AIDS, by neutralizing, removing or inhibiting different types of interferons, tumor necrosis factor, HLA class II antigens, IL6, and other pathologic factors and/or their receptors, as well as neutralizing, removing or inhibiting antitumor dies, including antibodies to a particular cell walls antigen. Treatment comprises administration of an anti-IL6 antibody, a neutralizing agent, or a combination thereof to the patient's blood or an immunosuppressing agent, or a combination thereof, to the patient's blood. The treatment of the patient's blood comprises a combined therapy involving extracorporeal immunosuppression, including with the administration of an agent immunosuppressant.

IN Skurkovich, Boris; Skurkovich, Simon V.

14 N Skurkovich, Boris; Skurkovich, Simon V.
15 AB The present disclosure concerns the treatment of a patient with autoimmune
16 disease, including AIDS, by neutralizing, removing or inhibiting
17 different types of interferons, tumor necrosis factor, HIV virus, II
18 antigens, IgE, and other pathologic factors and/or their receptors, as well
19 as neutralizing, removing or inhibiting autoantibodies, including
20 autoantibodies against target cells, T4 cells and NA. Treatment comprises
21 administration of at least one immune inhibitor, e.g. extracellular matrix, to
22 the patient's system, at least once, comprising an autoimmune
23 inhibitor, e.g. interferon, before or after the treatment of the patient with
24 comprising a combined therapy involving extracellular matrix and
25 in combination with the administration of an autoimmune inhibitor.

17 autoimmune inhibitor AIDS treatment extracellular
18 immunosorption
19 Fc receptors
20
21 RI: BSU (Biological study, unclassified); BICL (Biological study)
22 E, antibodies to; autoimmune inhibitors and extracellular
23 immunosorption for treatment of autoimmune diseases including
24 AIDS.

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17  Class II HLA antigens
    IgE
    Interferon .alpha.
    Interferon .alpha. receptors
    Interferon .gamma.
    Interferon .gamma. receptors
    Interleukin .alpha.
    Interleukin .alpha. receptors
    Tumor necrosis factor .alpha.
    Tumor necrosis factor .beta.
    Tumor necrosis factor .gamma.

```

ABSTRACT: Biological study, unclassified; EIDL (Biological study antibodies to aut immune inhibitors and extracorporeal immunosuppression for treatment of autoimmune diseases including AIDS

- 11. **AIDS** disease
- 12. **Ankylosing spondylitis**
- 13. **Antirheumatic drugs**
- 14. **Autoimmune diseases**
- 15. **Behcet's syndrome**
- 16. **Chen's disease**
- 17. **Drug delivery systems**
- 18. **Extracorporeal circulation**
- 19. **Immunosuppressants**
- 20. **Insulin dependent diabetes mellitus**
- 21. **Leptoty**
- 22. **Multiple sclerosis**
- 23. **Pneumonia arthritis**
- 24. **Schizophrenia**
- 25. **Systemic lupus erythematosus**
- 26. **Transplant rejection**
 - 27. **autoimmune inhibitors and extracorporeal immunosorption for treatment of autoimmune diseases including AIDS**

PL: BAC (Biological activity or effector, except adverse) ; THU
Therapeutic use ; AIDS: Biological study ; USSS (Uses:
autoimmune inhibitors and extracorporeal immunosorption for treatment
of autoimmune diseases including AIDS

Monoclonal antibodies
 Bi: BiN Biosynthetic preparation; THU Therapeutic use; BIL Biological study; FBS Preparation; CPE Uses
 anti-immune induction and extrinsic local immune system treatment
 Infectious diseases including AIDS

1 INA
 2 R11 R12 R13 R14 R15 R16 R17 R18 R19 R20 R21 R22 R23 R24 R25 R26 R27 R28 R29 R30 R31 R32 R33 R34 R35 R36 R37 R38 R39 R40 R41 R42 R43 R44 R45 R46 R47 R48 R49 R50 R51 R52 R53 R54 R55 R56 R57 R58 R59 R60 R61 R62 R63 R64 R65 R66 R67 R68 R69 R70 R71 R72 R73 R74 R75 R76 R77 R78 R79 R80 R81 R82 R83 R84 R85 R86 R87 R88 R89 R90 R91 R92 R93 R94 R95 R96 R97 R98 R99 R100
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[illegible][illegible][illegible]

2.2.2. *Antibodies*

AIDS

AIDS

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| INVENTOR: | ANDERSON, E. J. | PATENT INSTITUTE OF AMERICA |
| ASSIGNMENT NUMBER: | 177-0116 | AIRMAIL |
| SUBMITTER NUMBER: | 177-0116 | 1987-01-16 |
| TITLE: | Treatment of HIV infection by inhibiting AIDS virus replication, RNA processing, and integration | |
| INVENTOR: | Skurkovich, Simon V.; Skurkovich, Boris | |
| PATENT ASSIGNEE: | Advanced Biotechnology Concepts, Inc., USA | |
| ADDRESS: | U.S.A. | |
| COUNTRY: | USA | |
| LANGUAGE: | English | |
| FAMILY NO. COUNT: | 3 | |
| PATENT INFORMATION: | | |

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------------------|------|----------|-----------------|-------------|
| US 5626443 | A | 19970306 | US 1993-254346 | 19930220 |
| US 5648111 | A | 19970310 | US 1996-771831 | 19961223 |
| US 2000105511 | A1 | 20001109 | US 2001-785753 | 20011224 |
| FTH APPLN. INFO: | | | US 1993-254346 | A2 19931220 |
| | | | US 1996-771831 | A1 19961223 |
| | | | US 1997-995700 | A3 19971222 |

IN Skurkovich, Simon V.; Skurkovich, Boris

AB The present disclosure concerns a treatment for autoimmune diseases, including AIDS, by removing interferons, TNFs and receptors therefor, from a fluid. An extracorporeal device expresses fluids from a patient, including blood, plasma, cerebro spinal fluid, etc., to an immunosuppressant absorption removal. Following treatment, the fluid is returned to the patient. A patient with an extracorporeal device for removing pain, substances from a joint and spinal fluids is included.

immunosuppressive therapy, including TNF and receptor removal with extracorporeal immunoadsorption for treatment of autoimmune diseases, including AIDS

17 AIDS disease
 18 ascitic fluid
 19 Autoimmune diseases
 20 body fluid
 21 Cerebrospinal fluid
 22 Extracorporeal circulation
 23 Joint (anatomical)
 24 Plasma (blood)
 25 Synovial fluid
 26 interferon, TNF, and receptor removal with extracorporeal
 27 immunoabsorbent for treatment of autoimmune diseases, including
 28 AIDS

XX Antibodies

Monoclonal antibodies
for IEN, leishmaniasis, use ; TB^a, Therapeutic use ; B1-1, B1-2, anti-
cancer ; HIV^b, uses

Interferon, INF, and receptor-related with extra-synaptic
immunomodulation for treatment of autoimmune diseases, including

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11:  AIDS
12:  Antigens
13:  Interferon receptors
14:  Interferon alpha
15:  Interferon alpha receptors
16:  Interferon beta
17:  Interferon gamma
18:  Interferon gamma receptors
19:  Interferons
20:  HIV infection factors
21:  HIV infection factors
22:  HIV infection factors

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AIDS

Plasma apheresis, including plasmapheresis and double plasma exchange, is used for treatment of autoimmune diseases, including AIDS.

[illegible]

To examine a possible association between plasma viremia and interferon- α , IFN- α in patients with the acquired immunodeficiency syndrome (AIDS), we performed IFN plasma immunoadsorption by apheresis. IFN- α apheresis in four volunteers with AIDS who had sustained levels of endogenous plasma IFN- α . IFN- α apheresis with two plasma volume exchanges was performed daily for 5 days. Clinical signs and symptoms and hematologic, virologic, and immunologic parameters were monitored. Two subjects developed anemia from phlebotomy, and one had a catheter-associated bacteremia. The IFN- α apheresis was effective in transiently reducing IFN- α levels. In IFN- α apheresis, due to its rapid reformation, end-associated HIV-1 was unchanged, and three of four subjects had a subsequent increase in circulating plasma virus titer after the procedure. The recovery of HIV-1 titers after IFN- α apheresis and the effect of low-dose intravenous zalcitabine on the HIV-1 IFN- α showed characteristics on ELISA, Western blot, and indirect assays similar to the responses of the initial pretreatment. Reactivity, recruitment, and HIV-1-induced IFN- α s demonstrate nearly identical antiviral activities. The HIV-1 IFN- α eluted from the column was acid labile. The inability of large amounts of plasma IFN- α found in some patients with AIDS to affect viral burden likely reflects properties of the virus or of host factors independent of IFN- α .

A. J. Fischlauer, B. Stevens, M. Y. Liang, E. A. Reddy, R. Klein, P. Hain, M. J. Tsai, J. Malinich, S. Bellant, J. Skurkovich, S. Chakrabarti
AB To examine a possible association between plasma viremia and interferon- α , IFN- α in patients with the acquired immunodeficiency syndrome (AIDS), we performed IFN plasma immunoadsorption by apheresis. IFN- α apheresis in four volunteers with AIDS who had sustained levels of endogenous plasma IFN- α . IFN- α apheresis with two plasma volume exchanges was performed daily for 5 . . . from the column was not acid labile. The inability of large amounts of plasma IFN- α s found in some patients with AIDS to affect viral burden likely reflects properties of the virus or of host factors independent of IFN- α .

disturbances of interferon synthesis with the hyperproduction of unusual kinds of interferon may be the initial step which triggers an immune disease for which a chain of pathological reactions including the disturbance of several immunological and cytokine cascades, impaired regulation of the interferon may be a distinctive marker. In an immune condition the administration of interferon to animals or humans with an immune disease or an underlying or latent autoimmune condition can exacerbate or trigger the disease. Healthy people do not have interferon in their blood. This fundamental disturbance of interferon synthesis can result either from a genetic predisposition or from the influence of certain viruses or viral particles or both factors together.

AIDS has many features similar to autoimmune disease, including the hyperproduction of aberrant interferon, a type with restricted anti-HIV activity, protectively induced by HIV to allow its continued replication and survival. This interferon stimulates the production of certain cytokines and anti-antibodies which help unleash the potentially self-destructive powers of the immune system, bringing immunological chaos. In other words, while usual viruses induce normal interferon, which protects the cells against viral infection, HIV induces an abnormal, defective kind of interferon which ensures virus survival. Since there is no known effective method of destroying HIV directly, removing links in this chain of reactions which indirectly destroy HIV and possibly help best to improve the condition. **ABSTRACT TERMINATION** of HIV

Interferon synthesis with the hyperproduction of unusual kinds of interferon and triggers of immune disease and also a pathologic process. **AIDS** is the result of these into factors are interconnected.

[illegible]

1. ANSWER # 1: RI 111 11101 RI 111 111
 APLICATION NUMBER: 1111111111 RI 111
 2. FRONT NUMBER: 1111111111
 TITLE: METRIC 1111111111 AIDS AND 1111111111

AUTHOR: SKURKOVICH S; SKURKOVICH B
 TITLE: A METHOD FOR TREATING AIDS AND OTHER IMMUNE DEFICIENCIES AND IMMUNE DISORDERS.
 PATENT INFORMATION: US 4,244,222 Apr 1979
 SOURCE: Off. Gaz. U.S. Pat. Trademark Off., Pat., 1979 11:1 4, 2222.
 DOCUMENT TYPE: Patent
 FILE SEGMENT: SP; 111
 LANGUAGE: English
 CI: METHOD FOR TREATING AIDS AND OTHER IMMUNE DEFICIENCIES AND IMMUNE DISORDERS.
 AI: SKURKOVICH S; SKURKOVICH B

II. ANSWER 17 (P. 1) MEDICINE CONFIDENTIAL
 ABSTRACT NUMBER: 4244222 MEDICINE
 C. NUMBER: 4244222
 TITLE: A method for treating AIDS and other immune deficiencies and immune disorders.
 AUTHOR: Skurkovich S; Skurkovich B; Bellanti J A
 JOURNAL: JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 1979, 241, 10, 1000-1002
 PUB. COUNTRY: United States
 LANGUAGE: English
 FILE SEGMENT: SP; 111
 ENTRY NUMBER: 4244222
 ENTRY DATE: Entered 1979 10 10
 Last Edited: 1979 10 10
 Entered: 1979 10 10

AB This hypothesis is a presentation of a working model of the interferon (IFN) system as a cascade of sequentially interacting responses of IFN-alpha, -beta, and -gamma involve in modulation of the immune response. We propose that every antigen is an IFNogen. The first stage of immune responsiveness is associated primarily with the production of the family of IFN-alpha. In certain immunologically mediated diseases, including the autoimmune diseases and AIDS, disturbances in the synthesis of IFN-alpha occur with a switch to the production of predominantly acid-labile types, which have a negative immunoregulatory effect. Moreover, disturbances of IFN synthesis in the embryo or fetus can lead to deformities. Some viruses and other biological and chemical substances manifest a pathological effect by the IFN they induce. This IFN may help sustain the viruses and other substances which induce this IFN. We think it is unsafe to give patients immunoregulators in incomplete form. Thus, there is a potential danger in giving patients recombinant forms of IFNs and interleukin 2 produced in bacteria. In certain immune disorders, we may be able to treat patients by the binding or removal of hyperproduced IFNs from the body. This may lead to the restoration of immunologic balance and clinical improvement.

AI: Skurkovich S; Skurkovich B; Bellanti J A
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ALL LITERATURE AND ANSWER SENT APR 1981 AT 1 3 55
 SOURCE: V. N. H. 1111
 TIME IN U.S. LIBRARY: 11.11.11
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